09/777732

# WEST

## Freeform Search

**Database:** US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

**Term:** L4 and (monitor$3 near5 transplant$3)

**Display:** 10 **Documents in Display Format:** - **Starting with Number** 1

**Generate:** ○ **Hit List** ◉ **Hit Count** ○ **Side by Side** ○ **Image**

Search | Clear | Help | Logout | Interrupt

Main Menu | Show S Numbers | Edit S Numbers | Preferences | Cases

### Search History

**DATE: Wednesday, November 05, 2003** Printable Copy Create Case

| Set Name side by side | Query | Hit Count | Set Name result set |
|---|---|---|---|
| *DB=USPT,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ* | | | |
| L5 | L4 and (monitor$3 near5 transplant$3) | 5 | L5 |
| L4 | L3 and gene expression | 84 | L4 |
| L3 | l1 and (heme oxygenase or phophate dehydrogenase or cyclophilin or actin) | 152 | L3 |
| L2 | transplat$5 near5 kidney near5 (heme oxygenase or A20 or phosphate dehydrogenase or cyclophilin or actin) | 0 | L2 |
| L1 | transplant$5 near5 kidney | 2977 | L1 |

END OF SEARCH HISTORY

Generate Collection | Print

**Search Results** - Record(s) 1 through 5 of 5 returned.

☐ 1. 6514752 . 18 May 95; 04 Feb 03. Homologous recombination for universal donor cells and chimeric mammalian hosts. Kucherlapati; Raju, et al. 435/320.1; 435/325 435/455 536/23.1 800/13. C12N015/74 C12N005/02 C07H021/04 A01K067/033.

☑ 2. 6187534 . 24 Sep 97; 13 Feb 01. Methods of evaluating transplant rejection. Strom; Terry B., et al. 435/6; 435/7.24 536/24.31. C12Q001/68.

☐ 3. 6139835 . 18 May 95; 31 Oct 00. Homologous recombination for allogeneic donor cells. Kucherlapati; Raju, et al. 424/93.21; 435/320.1 435/325 435/455 435/463 514/44. C12N015/00 C12N015/63 C12N015/09.

☐ 4. 5574205 . 30 Dec 93; 12 Nov 96. Homologous recombination for universal donor cells and chimeric mammalian hosts. Kucherlapati; Raju, et al. 800/3; 424/9.2 424/93.21 435/320.1 800/11 800/18 800/22. C12N015/00 C12N005/00 A61K048/00 A61K049/00.

☐ 5. 5413923 . 11 Dec 92; 09 May 95. Homologous recombination for universal donor cells and chimeric mammalian hosts. Kucherlapati; Raju, et al. 435/463; 435/320.1 435/371. C12N015/00 C12N005/00.

Generate Collection | Print

| Term | Documents |
|---|---|
| MONITOR$3 | 0 |
| MONITOR | 488650 |
| MONITORA | 6 |
| MONITORAGE | 6 |
| MONITORALL | 1 |
| MONITORAND | 1 |
| MONITORATA | 1 |
| MONITORBLE | 1 |
| MONITORCRT | 1 |
| MONITORD | 11 |
| MONITORE | 21 |
| (L4 AND (MONITOR$3 NEAR5 TRANSPLANT$3)).USPT,JPAB,EPAB,DWPI. | 5 |

There are more results than shown above. Click here to view the entire set.

09777732

```
FILE 'HOME' ENTERED AT 11:08:44 ON 05 NOV 2003

=> file medline caplus biosis embase
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'MEDLINE' ENTERED AT 11:09:09 ON 05 NOV 2003

FILE 'CAPLUS' ENTERED AT 11:09:09 ON 05 NOV 2003
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2003 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'BIOSIS' ENTERED AT 11:09:09 ON 05 NOV 2003
COPYRIGHT (C) 2003 BIOLOGICAL ABSTRACTS INC.(R)

FILE 'EMBASE' ENTERED AT 11:09:09 ON 05 NOV 2003
COPYRIGHT (C) 2003 Elsevier Inc. All rights reserved.

=> s (heme oxygenase or A20)(10a)transpant#####
L1            0 (HEME OXYGENASE OR A20)(10A) TRANSPANT#####

=> s (heme oxygenase or A20)(10a) gene expression
L2          444 (HEME OXYGENASE OR A20)(10A) GENE EXPRESSION

=> s l2 and transplant#####
L3           16 L2 AND TRANSPLANT#####

=> s l3  and monitor###
L4            0 L3 AND MONITOR###

=> dup rem l3
PROCESSING COMPLETED FOR L3
L5           10 DUP REM L3 (6 DUPLICATES REMOVED)

=> d l5 1-10 bib ab kwic

L5   ANSWER 1 OF 10  CAPLUS  COPYRIGHT 2003 ACS on STN
AN   2003:532744  CAPLUS
DN   139:96408
TI   Biliverdin reductase modulation of heme oxygenase-1
     (HO-1) gene expression and methods for treating
     HO-1-mediated conditions
IN   Maines, Mahin D.
PA   University of Rochester, USA
SO   PCT Int. Appl., 51 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.         KIND  DATE           APPLICATION NO.  DATE
     ---------------    ----  --------       ---------------  --------
PI   WO 2003055981      A2    20030710       WO 2002-US41167  20021220
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ,
             UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD,
             RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG,
             CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,
```

MR, NE, SN, TD, TG
PRAI US 2001-342247P P 20011221
AB A method of modifying HO-1 transcription is disclosed. The method includes modifying the nuclear concn. of biliverdin reductase, or fragments or variants thereof which bind to heme oxygenase-1 gene regulatory sequence AP-1 in a cell, whereby increased nuclear biliverdin reductase levels increases HO-1 transcription and a decrease decreases transcription of HO-1. Biliverdin reductase-mediated modulation of HO-1 gene expression may be used to treat various HO-1-assocd. disorders and diseases. Thus, human biliverdin reductase was shown to dimerize and bind to AP-1 sites in the HO-1 gene promoter. Mutations in the leucine zipper domains abolished this binding. In COS cells transfected with antisense biliverdin reductase RNA, the increase of HO-1 mRNA levels to menadione exposure was inhibited.
TI Biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions
IT Genetic element
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(AP-1 site, biliverdin reductase binding to; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(BKB1R, biliverdin reductase regulation of expression of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HO-1, biliverdin reductase regulation of expression of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ICR-17, biliverdin reductase regulation of expression of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Ier5, biliverdin reductase regulation of expression of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MCP-1, biliverdin reductase regulation of expression of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Abrasion
Asthma
Athlete's foot
Burn
Human
Immunosuppression
Inflammation
Skin, disease
**Transplant** rejection
(biliverdin reductase modulation of **hem oxygenase** -1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Inflammation
(chronic; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Mucous membrane
(disease, ulcerations of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Mouth
(disorder; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Lung
(epithelium, hyperoxia in; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Embryo, animal
(fetus, growth of, problems of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Blood vessel
(high resistance disorders of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Eye, disease
(hypoxia-assocd.; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Drug delivery systems
(liposomes, biliverdin reductase-contg., therapeutic use of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Artery, disease
(restenosis; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Hypotension
(sepsis-assocd.; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Antisense RNA
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(to biliverdin reductase nucleic acid; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT Gene therapy
(to modulate biliverdin reductase levels; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT 9059-22-7, **Heme oxygenase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(biliverdin reductase modulation of **heme oxygenase** -1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT 9074-10-6, Biliverdin reductase
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(biliverdin reductase modulation of **heme oxyg nase** -1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)
IT 635-65-4, Bilirubin, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(hyperbilirubinemia; biliverdin reductase modulation of **hem**

**oxygenase**-1 (HO-1) **gene expr ssion** and
methods for treating HO-1-mediated conditions)

IT 557809-65-1 557809-66-2 557809-67-3 557809-68-4 557809-69-5
557809-70-8 557809-71-9 557809-72-0 557809-73-1 557809-74-2
557809-75-3 557809-76-4 557809-77-5 557809-78-6 557809-79-7
557809-80-0 557809-81-1 557809-82-2 557809-83-3 557809-84-4
557809-85-5 557809-86-6 557809-87-7 557809-88-8 557809-89-9
557809-90-2 557809-91-3 557809-92-4 557809-93-5 557809-94-6
557809-95-7 557809-96-8 557809-97-9 557809-98-0 557809-99-1
557810-00-1 557810-01-2 557810-02-3
RL: PRP (Properties)
(unclaimed sequence; biliverdin reductase modulation of **heme**
**oxygenase**-1 (HO-1) **gene expression** and
methods for treating HO-1-mediated conditions)

L5 ANSWER 2 OF 10 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 2003305664 EMBASE
TI Accommodation in ABO-incompatible kidney allografts, a novel mechanism of
self-protection against antibody-mediated injury.
AU Park W.D.; Grande J.P.; Ninova D.; Nath K.A.; Platt J.L.; Gloor J.M.;
Stegall M.D.
CS M.D. Stegall, Department of Surgery, Mayo Clinic, Rochester, MN, United
States. stegall.mark@mayo.edu
SO American Journal of Transplantation, (2003) 3/8 (952-960).
Refs: 42
ISSN: 1600-6135 CODEN: AJTMBR
CY Denmark
DT Journal; Article
FS 026 Immunology, Serology and Transplantation
037 Drug Literature Index
LA English
SL English
AB To elucidate the mechanism of self-protection against anti-donor
blood-group antibody known as accommodation, we studied 16 human
ABO-incompatible living-donor kidney **transplant** recipients at 3
and 12 months post **transplantation**. Both circulating
anti-blood-group antibody and the target blood-group antigen in the graft
were demonstrable in all patients after **transplantation**.
Thirteen of 16 grafts had normal renal function and histology, while three
grafts with prior humoral rejection demonstrated significant
glomerulopathy and thus did not meet the criterion for accommodation.
Using microarrays, we compared five 1-year protocol ABO-compatible renal
graft biopsies to four accommodated ABO-incompatible graft biopsies.
Significant alterations in gene expression in 440 probe sets, including
SMADs, protein tyrosine kinases, TNF-.alpha. and Mucin 1 were identified.
We verified these changes in **gene expression** using
RT-PCR and immunohistochemistry. **Heme oxygenase**-1,
Bcl-2 and Bcl-xl were not increased in ABO-incompatible grafts at any
time-point. We conclude that accommodation is always present in
well-functioning, long-surviving ABO-incompatible kidney
**transplants**. This self-protection against antibody-mediated damage
may involve several novel mechanisms including the disruption of normal
signal transduction, attenuation of cellular adhesion and the prevention
of apoptosis.
AB To elucidate the mechanism of self-protection against anti-donor
blood-group antibody known as accommodation, we studied 16 human
ABO-incompatible living-donor kidney **transplant** recipients at 3
and 12 months post **transplantation**. Both circulating
anti-blood-group antibody and the target blood-group antigen in the graft
were demonstrable in all patients after **transplantation**.
Thirteen of 16 grafts had normal renal function and histology, while three
grafts with prior humoral rejection demonstrated significant
glomerulopathy. . . in 440 probe sets, including SMADs, protein

tyrosine kinases, TNF-.alpha. and Mucin 1 were identified. We verified these changes in **g ne expression** using RT-PCR and immunohistochemistry. **Heme oxygenase**-1, Bcl-2 and Bcl-xl were not increased in ABO-incompatible grafts at any time-point. We conclude that accommodation is always present in well-functioning, long-surviving ABO-incompatible kidney **transplants**. This self-protection against antibody-mediated damage may involve several novel mechanisms including the disruption of normal signal transduction, attenuation of cellular. . .

CT Medical Descriptors:
***transplantation tolerance**
*blood group ABO incompatibility
*kidney allograft
*immune mediated injury
postoperative period
kidney function
histology
humoral immunity
graft rejection
glomerulopathy
DNA microarray
kidney biopsy
gene expression
reverse transcription polymerase chain reaction
immunohistochemistry
signal transduction
cell adhesion
apoptosis
human
clinical. . .

L5 ANSWER 3 OF 10 MEDLINE on STN DUPLICATE 1
AN 2002491056 MEDLINE
DN 22239063 PubMed ID: 12352326
TI Oxidative stress in kidney **transplant** patients with calcineurin inhibitor-induced hypertension: effect of ramipril.
AU Calo Lorenzo A; Davis Paul A; Giacon Bruno; Pagnin Elisa; Sartori Michelangelo; Riegler Peter; Antonello Augusto; Huber Walter; Semplicini Andrea
CS Department of Clinical and Exprimental Medicine, Clinica Medica 4, University of Padova, Padova, Italy.. renzcalo@unipd.it
SO JOURNAL OF CARDIOVASCULAR PHARMACOLOGY, (2002 Oct) 40 (4) 625-31. Journal code: 7902492. ISSN: 0160-2446.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200303
ED Entered STN: 20020928
Last Updated on STN: 20030306
Entered Medline: 20030305
AB In patients with cyclosporine-induced hypertension, upregulation of the nitric oxide system and oxidative stress were shown, which could induce hypertension, remodeling, and chronic rejection by increasing nitric oxide catabolism. However, it is still debated whether cyclosporine and tacrolimus exert a different action. The aim of the current study was to compare the effects of cyclosporine and tacrolimus on markers of oxidative stress and endothelial dysfunction in kidney **transplant** patients with posttransplant hypertension. Monocyte p22, a NADH/NADPH system subunit, transforming growth factor-beta (TGF-beta), **hem oxygenase**-1 (HO-1), and endothelial NOS **gene expression** were measured in 16 patients. Angiotensin II is a potent stimulator of oxidative stress and angiotensin-converting enzyme inhibition may blunt this effect. Therefore, the same parameters were

measured before and after 2 months of treatment with ramipril (5 mg/d). At baseline, in cyclosporine-and tacrolimus-treated patients, p22 and TGF-beta mRNA were similarly increased in comparison with normotensive healthy controls (0.90 +/- 0.05 d.u. and 0.83 +/- 0.05 in cyclosporine, 0.89 +/- 0.07 and 0.84 +/- 0.05 in tacrolimus; 0.53 +/- 0.07 and 0.75 +/- 0.03 in controls, respectively; p < 0.001). Endothelial NOS mRNA was increased in cyclosporine-and tacrolimus-treated patients in comparison with controls (0.92 +/- 0.09, 0.96 +/- 0.04, and 0.37 +/- 0.05 respectively; p < 0.001), whereas no difference was found between patients and controls in HO-1 mRNA. Ramipril reduced blood pressure (from 140 +/- 11/91 +/- 7 mm Hg to 129 +/- 6/85 +/- 5 mm Hg in cyclosporine and from 138 +/- 7/92 +/- 7 mm Hg to 127 +/- 10/82 +/- 6 mm Hg in tacrolimus group; p < 0.02 with no difference between groups). Ramipril also reduced p22 (to 0.83 +/- 0.05 in cyclosporine, p < 0.03 and to 0.81 +/- 0.08 in tacrolimus; p < 0.01) and TGF-beta mRNA (to 0.72 +/- 01 in cyclosporine, p < 0.02, and to 0.73 +/- 0.05 in tacrolimus; p < 0.01) with no difference between groups, but it did not change HO-1 and ecNOS mRNA. Cyclosporine and tacrolimus induce a comparable oxidative stress in kidney **transplant** patients with posttransplant hypertension. The association of ramipril normalizes blood pressure and reduces the oxidative stress induced by both drugs.

TI Oxidative stress in kidney **transplant** patients with calcineurin inhibitor-induced hypertension: effect of ramipril.

AB . . . study was to compare the effects of cyclosporine and tacrolimus on markers of oxidative stress and endothelial dysfunction in kidney **transplant** patients with posttransplant hypertension. Monocyte p22, a NADH/NADPH system subunit, transforming growth factor-beta (TGF-beta), **heme oxygenase**-1 (HO-1), and endothelial NOS **gene expression** were measured in 16 patients. Angiotensin II is a potent stimulator of oxidative stress and angiotensin-converting enzyme inhibition may blunt. . . groups, but it did not change HO-1 and ecNOS mRNA. Cyclosporine and tacrolimus induce a comparable oxidative stress in kidney **transplant** patients with posttransplant hypertension. The association of ramipril normalizes blood pressure and reduces the oxidative stress induced by both drugs.

CT . . .

Hypertension: CI, chemically induced
*Hypertension: DT, drug therapy
Hypertension: ME, metabolism
Immunosuppressive Agents: AE, adverse effects
Immunosuppressive Agents: PD, pharmacology
***Kidney Transplantation**
Middle Age
Monocytes: DE, drug effects
Monocytes: ME, metabolism
*Oxidative Stress: DE, drug effects
Oxidative Stress: PH, physiology
*Ramipril:. . .

L5 ANSWER 4 OF 10 MEDLINE on STN DUPLICATE 2
AN 2002412392 MEDLINE
DN 22157033 PubMed ID: 12166345
TI Increased **heme oxygenase**-1 **gene expression** in the livers of patients with portal hypertension due to severe hepatic cirrhosis.
AU Matsumi M; Takahashi T; Fujii H; Ohashi I; Kaku R; Nakatsuka H; Shimizu H; Morita K; Hirakawa M; Inagaki M; Sadamori H; Yagi T; Tanaka N; Akagi R
CS Department of Anaesthesiology and Resuscitation, Okayama University Medical School, Okayama, Japan.
SO JOURNAL OF INTERNATIONAL MEDICAL RESEARCH, (2002 May-Jun) 30 (3) 282-8. Journal code: 0346411. ISSN: 0300-0605.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals
EM 200301
ED Entered STN: 20020809
Last Updated on STN: 20030117
Entered Medline: 20030116
AB Surgical bleeding associated with splanchnic hyperaemia due to portal hypertension complicates the anaesthetic management of hepatic **transplantation**. Although the mechanism(s) of portal hypertension are not fully understood, carbon monoxide, a product of the heme oxygenase (HO) reaction, is thought to be one of the endogenous vasodilators in the liver. In this study, the expression of mRNA encoding inducible HO isozyme (HO-1) in the livers of patients with portal hypertension undergoing hepatic **transplantation** was determined in comparison with those without portal hypertension. HO-1 mRNA levels were significantly greater in the portal hypertension group than in the group without portal hypertension. In contrast with HO-1, the gene expression of non-specific delta-amino-levulinate synthase (ALAS-N), which is down-regulated by heme in the liver, was the same in both groups. These results suggest that HO-1 is up-regulated through heme-independent stimuli according to the development of portal hypertension, and that induced HO-1 plays a pathophysiological role in portal hypertension through carbon monoxide production.
TI Increased **heme oxygenase**-1 **gene expression** in the livers of patients with portal hypertension due to severe hepatic cirrhosis.
AB Surgical bleeding associated with splanchnic hyperaemia due to portal hypertension complicates the anaesthetic management of hepatic **transplantation**. Although the mechanism(s) of portal hypertension are not fully understood, carbon monoxide, a product of the heme oxygenase (HO) reaction,. . . study, the expression of mRNA encoding inducible HO isozyme (HO-1) in the livers of patients with portal hypertension undergoing hepatic **transplantation** was determined in comparison with those without portal hypertension. HO-1 mRNA levels were significantly greater in the portal hypertension group. . .

L5 ANSWER 5 OF 10 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:617958 BIOSIS
DN PREV200200617958
TI Upregulation of **heme oxygenase**-1 **gene expression** upon reperfusion of human liver **transplants** is associated with decreased ischemia/reperfusion injury.
AU Geuken, Erwin [Reprint author]; Visser, Dorien S. [Reprint author]; Moshage, Han M. [Reprint author]; de Jong, Koert P. [Reprint author]; Peeters, Paul M. [Reprint author]; Leuvenink, Henri M. [Reprint author]; Jansen, Peter L. [Reprint author]; Slooff, Maarten J. [Reprint author]; Porte, Robert J. [Reprint author]
CS University Medical Center Groningen, Groningen, Netherlands
SO Hepatology, (October, 2002) Vol. 36, No. 4 Part 2, pp. 201A. print.
Meeting Info.: 53rd Annual Meeting on the Liver. BOSTON, MA, USA. November 01-05, 2002.
CODEN: HPTLD9. ISSN: 0270-9139.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 4 Dec 2002
Last Updated on STN: 4 Dec 2002
TI Upregulation of **heme oxygenase**-1 **gene expression** upon reperfusion of human liver **transplants** is associated with decreased ischemia/reperfusion injury.
IT . . .
and Molecular Biophysics)
IT Diseases
ischemia-reperfusion injury: injury, vascular disease
Reperfusion Injury (MeSH)

IT Chemicals & Biochemicals
alanine aminotransferase; aspartate aminotransferase; **heme oxygenase**-1: **gene expression**
IT Methods & Equipment
liver **transplantation**: surgical method
IT Miscellaneous Descriptors
Meeting Abstract

L5 ANSWER 6 OF 10 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:360707 BIOSIS
DN PREV200100360707
TI CsA or FK-506 induced post-**transplant** hypertension and oxidative stress in kidney **transplanted** patients. Effect of ramipril.
AU Calo, Lorenzo [Reprint author]; Giacon, Bruno; Pagnin, Elisa; Sartori, Michelangelo; Huber, Walter; Semplicini, Andrea
CS Clin. Exp. Med., Univ. Padova, Padova, Italy
SO American Journal of Hypertension, (April, 2001) Vol. 14, No. 4 Part 2, pp. 252A. print.
Meeting Info.: Sixteenth Annual Scientific Meeting of the American Society of Hypertension. San Francisco, California, USA. May 15-19, 2001. American Society of Hypertension.
CODEN: AJHYE6. ISSN: 0895-7061.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 2 Aug 2001
Last Updated on STN: 19 Feb 2002
TI CsA or FK-506 induced post-**transplant** hypertension and oxidative stress in kidney **transplanted** patients. Effect of ramipril.
IT . . .
system, graft; monocyte: blood and lymphatics, immune system
IT Diseases
endothelial dysfunction: vascular disease
Endothelium, Vascular: PP, physiopathology (MeSH)
IT Diseases
post-**transplant** hypertension: toxicity, vascular disease, treatment
Hypertension (MeSH)
IT Chemicals & Biochemicals
CsA [cyclosporin A]: immunosuppressant-drug, pharmacodynamics, toxicity; FK-506: immunosuppressant-drug, pharmacodynamics, toxicity;. . .
IT Methods & Equipment
RT-PCR [reverse transcriptase-polymerase chain reaction]: diagnostic method, polymerase chain reaction; kidney **transplantation**: therapeutic method
IT Miscellaneous Descriptors
nitric oxide system; oxidative stress; Meeting Abstract
GEN HO-1 gene [**heme oxygenase**-1 **gene**]: **xpression**; TGF-beta gene: expression; ecNOS gene [endothelial nitric oxide synthase gene]: expression; p22-phox gene: expression

L5 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2003 ACS on STN
AN 2000:138823 CAPLUS
DN 133:56709
TI Expression of heme oxygenase-1 by endothelial cells: a protective response to injury in **transplantation**
AU Soares, M. P.; Brouard, S.; Smith, R. N.; Otterbein, L.; Choi, A. M.; Bach, F. H.
CS Immunobiology Research Center, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, 02215, USA
SO Emerging Therapeutic Targets (2000), 4(1), 11-27
CODEN: ETTAF7; ISSN: 1460-0412
PB Ashley Publications

DT Journal; General Review
LA English
AB A review, with 131 refs. Endothelial cells (EC) play a pivotal role in the regulation of inflammation by expressing a series of pro- and anti-inflammatory genes that are assocd. with the activation of these cells. The nature of these genes and the regulation of their expression may be particularly important for the outcome of immediately vascularised **transplants**. We refer to the set of anti-inflammatory genes that are expressed during EC activation as protective genes because they can block the expression of pro-inflammatory genes assocd. with EC activation and prevent EC apoptosis. In this review we discuss data that supports the hypothesis that expression of these protective genes in a **transplanted** organ can promote its survival. We will focus on the description of one such protective gene, heme oxygenase-1 (HO-1). The first part of the review discusses the potential role of EC activation in regulating inflammatory responses such as those assocd. with the rejection of **transplanted** organs. The second part discusses the mol. mechanisms that regulate the expression of HO-1 in EC as well as the mol. mechanism by which the expression of this gene can regulate EC activation. The third part discusses potential mechanisms by which HO-1 may contribute to suppress different phases of the rejection of **transplanted** organs, e.g., ischemia reperfusion injury, acute rejection and chronic failure. In the last part we discuss the role of HO-1 in establishing long-term survival of organs that are **transplanted** across different species, an approach referred to as xenotransplantation.
RE.CNT 131 THERE ARE 131 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI Expression of heme oxygenase-1 by endothelial cells: a protective response to injury in **transplantation**
AB A review, with 131 refs. Endothelial cells (EC) play a pivotal role in the regulation of inflammation by expressing a series of pro- and anti-inflammatory genes that are assocd. with the activation of these cells. The nature of these genes and the regulation of their expression may be particularly important for the outcome of immediately vascularised **transplants**. We refer to the set of anti-inflammatory genes that are expressed during EC activation as protective genes because they can block the expression of pro-inflammatory genes assocd. with EC activation and prevent EC apoptosis. In this review we discuss data that supports the hypothesis that expression of these protective genes in a **transplanted** organ can promote its survival. We will focus on the description of one such protective gene, heme oxygenase-1 (HO-1). The first part of the review discusses the potential role of EC activation in regulating inflammatory responses such as those assocd. with the rejection of **transplanted** organs. The second part discusses the mol. mechanisms that regulate the expression of HO-1 in EC as well as the mol. mechanism by which the expression of this gene can regulate EC activation. The third part discusses potential mechanisms by which HO-1 may contribute to suppress different phases of the rejection of **transplanted** organs, e.g., ischemia reperfusion injury, acute rejection and chronic failure. In the last part we discuss the role of HO-1 in establishing long-term survival of organs that are **transplanted** across different species, an approach referred to as xenotransplantation.
ST review endothelium organ **transplant** rejection heme oxygenase
IT Gene, animal
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(HO-1; **heme oxygenase**-1 **gene**
**expression** in endothelial cells as protective response to injury in **transplantation**)
IT Blood vessel
(endothelium; **heme oxygenas** -1 **gene**
**xpression** in endothelial cells as protective response to injury in **transplantation**)

IT **Transplant** and **Transplantation**
**Transplant** rejection
(**heme oxygenase**-1 **gene expression** in endothelial cells as protective response to injury in **transplantation**)
IT Reperfusion
(injury; **heme oxygenase**-1 **gene expression** in endothelial cells as protective response to injury in **transplantation**)
IT **Transplant** and **Transplantation**
(xenotransplant; **heme oxygenase**-1 **gene expression** in endothelial cells as protective response to injury in **transplantation**)
IT 9059-22-7
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(1; **heme oxygenase**-1 **gene expression** in endothelial cells as protective response to injury in **transplantation**)
IT 124-38-9, Carbon dioxide, biological studies 635-65-4, Bilirubin, biological studies 7439-89-6, Iron, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**heme oxygenase**-1 **gene expression** in endothelial cells as protective response to injury in **transplantation**)

L5 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2003 ACS on STN
AN 1999:194175 CAPLUS
DN 130:236480
TI Characterization of APRIL growth factor
IN Tschopp, Jurg
PA Biogen, Inc., USA
SO PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|---|
| PI | WO 9912965 | A2 | 19990318 | WO 1998-US19191 | 19980911 |
| | WO 9912965 | A3 | 19990603 | | |
| | W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | |
| | RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG | | | | |
| | CA 2303615 | AA | 19990318 | CA 1998-2303615 | 19980911 |
| | AU 9893162 | A1 | 19990329 | AU 1998-93162 | 19980911 |
| | AU 759717 | B2 | 20030417 | | |
| | EP 1027431 | A2 | 20000816 | EP 1998-946066 | 19980911 |
| | R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO | | | | |
| | BR 9812634 | A | 20000822 | BR 1998-12634 | 19980911 |
| | EE 200000147 | A | 20010215 | EE 2000-200000147 | 19980911 |
| | JP 2001515712 | T2 | 20010925 | JP 2000-510770 | 19980911 |
| | NZ 503850 | A | 20021220 | NZ 1998-503850 | 19980911 |
| | NO 2000001242 | A | 20000511 | NO 2000-1242 | 20000309 |
| | MX 200002407 | A | 20001030 | MX 2000-2407 | 20000309 |
| | US 2003138884 | A1 | 20030724 | US 2002-138073 | 20020501 |
| PRAI | US 1997-58786P | P | 19970912 | | |

US 1998-79384P P 19980326
WO 1998-US19191 W 19980911
US 2000-520489 A3 20000308

AB The author discloses the nucleic acid and protein sequences for human and mouse APRIL growth factor (A Proliferation Inducing Ligand), a novel member of the tumor necrosis factor family. Gene expression is demonstrated in normal and malignant tissue and numerous tumor cell lines. In addn., APRIL is shown to be mitogenic for T lymphocytes (Jurkat) and B lymphocytes (Raji).

IT Animal cell line
(**A20**; **gene expression** for APRIL growth factor in)

IT Autoimmune disease
**Transplant** rejection
(APRIL for treatment of)

L5 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2003 ACS on STN
AN 2000:15330 CAPLUS
DN 132:288573
TI Heme oxygenase-1 overexpression protects genetically fat Zucker rat livers from ischemia/reperfusion injury
AU Amersi, Farin; Buelow, Roland; Farmer, Douglas; Kato, Hirohisa; Ke, Bibo; Ghobrial, Mark; Busuttil, Ronald W.; Kupiec-Weglinski, Jerzy W.
CS Dumont-UCLA Transplant Center, Division of Liver and Pancreas Transplantation, Department of Surgery, School of Medicine, University of California, Los Angeles, CA, USA
SO Surgical Forum (1999), 50, 385-387
CODEN: SUFOAX; ISSN: 0071-8041
PB American College of Surgeons
DT Journal
LA English
AB Systemic pretreatment with cobalt protoporphyrin (CoPP) or local adenoviral heme oxygenase-1 (HO-1) gene transfer equally protected against ischemia/reperfusion injury in the livers of Zucker rats (rats with steatotic livers) in an ex vivo model of cold ischemia. Pretreatment with CoPP greatly improved the survival rate after reperfusion orthotopic liver **transplantation** (OLT) of cold ischemia-subjected fatty livers into lean Zucker rats. This is the first report to document the utility of HO-1 in increasing the donor pool through modulation of marginal steatotic livers.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB Systemic pretreatment with cobalt protoporphyrin (CoPP) or local adenoviral heme oxygenase-1 (HO-1) gene transfer equally protected against ischemia/reperfusion injury in the livers of Zucker rats (rats with steatotic livers) in an ex vivo model of cold ischemia. Pretreatment with CoPP greatly improved the survival rate after reperfusion orthotopic liver **transplantation** (OLT) of cold ischemia-subjected fatty livers into lean Zucker rats. This is the first report to document the utility of HO-1 in increasing the donor pool through modulation of marginal steatotic livers.
ST **heme oxygenase gene expression**
steatotic liver **transplantation** success
IT Gene, animal
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(HO-1; heme oxygenase-1 gene overexpression improves steatotic liver **transplantation** success)
IT Temperature effects, biological
(cold; heme oxygenase-1 gene overexpression improves steatotic liver **transplantation** success)
IT Liver, disease
(fatty; heme oxygenase-1 gene overexpression improves steatotic liver **transplantation** success)

IT Gene therapy
Organ preservation
Reperfusion
(heme oxygenase-1 gene overexpression improves steatotic liver **transplantation** success)
IT Reperfusion
(injury, of ischemic, steatotic liver **transplant**; heme oxygenase-1 gene overexpression improves steatotic liver **transplantation** success)
IT Liver, disease
(injury, of steatotic liver **transplant**; heme oxygenase-1 gene overexpression improves steatotic liver **transplantation** success)
IT Liver, disease
(ischemia, cold; heme oxygenase-1 gene overexpression improves steatotic liver **transplantation** success)
IT **Transplant** and **Transplantation**
**Transplant** and **Transplantation**
(liver, orthotopic; heme oxygenase-1 gene overexpression improves steatotic liver **transplantation** success)
IT Liver
Liver
(**transplant**, orthotopic; heme oxygenase-1 gene overexpression improves steatotic liver **transplantation** success)
IT 9059-22-7
RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(1, gene; heme oxygenase-1 gene overexpression improves steatotic liver **transplantation** success)
IT 14325-03-2
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(heme oxygenase-1 gene overexpression improves steatotic liver **transplantation** success)

L5 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2003 ACS on STN
AN 1997:94851 CAPLUS
DN 126:170280
TI Accommodation of vascularized xenografts: Expression of "protective genes" by donor endothelial cells in a host Th2 cytokine environment
AU Bach, Fritz H.; Ferran, Christiane; Hechenleitner, Paul; Mark, Walter; Koyamada, Nozomi; Miyatake, Tsukasa; Winkler, Hans; Badrichani, Anne; Candinas, Daniel; Hancock, Wayne W.
CS Dep. Surgery, New England Deaconess Hosp. and Harvard Medical School, Boston, MA, 02215, USA
SO Nature Medicine (New York) (1997), 3(2), 196-204
CODEN: NAMEFI; ISSN: 1078-8956
PB Nature Publishing Co.
DT Journal
LA English
AB Organ xenografts under certain circumstances survive in the presence of anti-graft antibodies and complement, a situation referred to as "accommodation". The authors find that the endothelial cells (ECs) in hamster hearts that accommodate themselves in rats express genes, such as A20 and bcl-2, that in vitro protect ECs from apoptosis and prevent upregulation in those cells of proinflammatory genes such as cytokines, procoagulant and adhesion mols. Hearts that are rejected do not express these genes. In addn., vessels of rejected hearts show florid **transplant** arteriosclerosis whereas those of accommodated hearts do not. Accommodated xenografts have an ongoing T helper cell type 2 (Th2) cytokine immune response, whereas the rejected grafts have a Th1 response. The authors propose a model for factors that contribute to the survival of xenografts and the avoidance of **transplant**

arteriosclerosis.

AB Organ xenografts under certain circumstances survive in the presence of anti-graft antibodies and complement, a situation referred to as "accommodation". The authors find that the endothelial cells (ECs) in hamster hearts that accommodate themselves in rats express genes, such as A20 and bcl-2, that in vitro protect ECs from apoptosis and prevent upregulation in those cells of proinflammatory genes such as cytokines, procoagulant and adhesion mols. Hearts that are rejected do not express these genes. In addn., vessels of rejected hearts show florid **transplant** arteriosclerosis whereas those of accommodated hearts do not. Accommodated xenografts have an ongoing T helper cell type 2 (Th2) cytokine immune response, whereas the rejected grafts have a Th1 response. The authors propose a model for factors that contribute to the survival of xenografts and the avoidance of **transplant** arteriosclerosis.

IT Gene, animal
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**A20**; donor endothelium **gene expression** and helper T-cell infiltration in relation to accommodation of vascularized xenografts)

IT Proteins, specific or class
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(gene **A20**; accommodation of vascularized xenografts in relation to donor endothelium **gene expression** for)

IT Arteriosclerosis
(**transplant**; donor endothelium gene expression and helper T-cell infiltration in relation to accommodation of vascularized xenografts)

IT **Transplant** and **Transplantation**
**Transplant** and **Transplantation**
(xenotransplant, heart; donor endothelium gene expression and helper T-cell infiltration in relation to accommodation of vascularized xenografts)

IT 9059-22-7, **Heme oxygenase**
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(1; accommodation of vascularized xenografts in relation to donor endothelium **gene expression** for)

=>

```
> s strom.in.
L6          411 STROM.IN.

=> s l6 and (heme oxygenase or A20)
L7            0 L6 AND (HEME OXYGENASE OR A20)

=> s l6 and transplant#####
L8           12 L6 AND TRANSPLANT#####

=> s l8 and (oxygenase or A20)
L9            0 L8 AND (OXYGENASE OR A20)

=>
```